LA GESTIONE DEL PAZIENTE ANEMICO IN PRONTO SOCCORSO: FERRO VS SANGUE

L’anemia serve!

Pasquale, 88 anni, circa 70 kg.

- K vescicale con multiple localizzazioni secondarie
- IRC di grado severo (GFR 19 sec C&G)
- Iponatremia
- CHF III NYHA

Quick-look: spaventato, pallido, marcatamente astenico

A: vie aeree pervie

B: crepitazioni alle basi BR 20/minuto SaO_2 93% con FiO_2 21%

C: soffio olosistolico sulla punta 4/6 Levine PA 150-75 mm/Hg

D: CGS 15

E: NTC 36.6 °C NRS 8

Eco-torace B-profile

CHIMICA CLINICA - IMMUNOMETRIA

PASQUALE

Cod. struttura 470158

Data di nascita : 22/02/1928
(3317) (30) (*)

NAPOLI, prelievo del 02/03/2016 Pag. 1 di 1

determinazioni richieste	esiti		valori di riferimento (Uomo)
AZOTEMIA Metodo cinetico Ureasi-GLDH	71	mg/dl	10 - 50
CREATININA Metodo cinetico Jaffè	2,65	mg/dl	0,7 - 1,3
RETICOLOCITI	3,3	%	0,5 - 2,5
SIDEREMIA Metodo colorimetrico con Ferene senza deproteinizzazione	13	µg/dL	49 - 181
FERRITINA Metodo chemiluminescenza	602,6	ng/ml	22 - 322
TRANSFERRINEMIA Nefelometria	103	mg/dl	200 - 360

Il Direttore Tecnico
Dott. Claudia Marengo

VIA TINO DI CAMAINO, 6 (Piazza Medaglie d'Oro) - 80129 NAPOLI - TEL. 081.5781507 PBX - E-mail: semeios@libero.it
I RISULTATI DELLE ANALISI SONO SOTTOPOSTI A CONTROLLI INTERNI A TITOLO NOTO E V.E.Q. (VALUTAZIONE ESTERNA DELLA QUALITÀ)

Ora: 15.45
rtato il: 02/03/2016 Ora: 16.30

SAME	RISULTATO			
ESAME EMOCROMOCITOMETRICO				
3C	12,0 *	$10^3/mm^3$	4.2 - 10.5	
C	* 2,72	$10^6/mm^3$	4.0 - 5.8	
iB	* 7,6	g/dl	11.5 -17.5	LYN
T	* 23,6	%	36.0 - 55.0	MON
:V	87,0	μm^3	78,0 - 98,0	NEU
:H	27,8	pg	27,0 - 34,0	EOS
:HC	32,1	g/dl	32,0 - 36,0	BAS
W	16,7	%	11,0 - 18,0	
T	516 *	$10^3/mm^3$	150 - 400	

- PCR 3.7
- Lact 2.9
- HGT 211

"L'oncologo ha detto che deve fare la trasfusione!!!"

Benefits and harms of red blood cell transfusions in patients with septic shock in the Intensive Care Unit

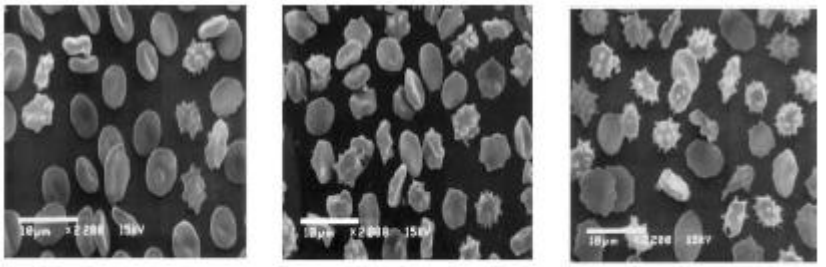
Risks of anaemia

- Low DO_2
- Ischemia
- Organ dysfunction
- Multiple organ dysfunction

Risks of transfusion

- Infectious
- Non-infectious
  - Storage-lesion
  - Immunomodulation
- Cost

Figure 2 Showing risks to be outweighted before decision to transfuse.



5th day of storage | 14th day of storage | 42nd day of storage

Figure 1 Electron microscope images showing corpuscular changes in red blood cells during storage.34

Per gentile concessione di Mario Rugna

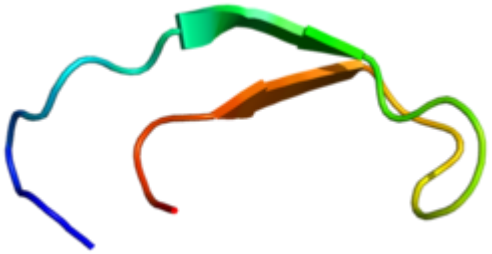
Published in final edited form as:
Nat Rev Immunol. 2015 August ; 15(8): 500–510. doi:10.1038/nri3863.

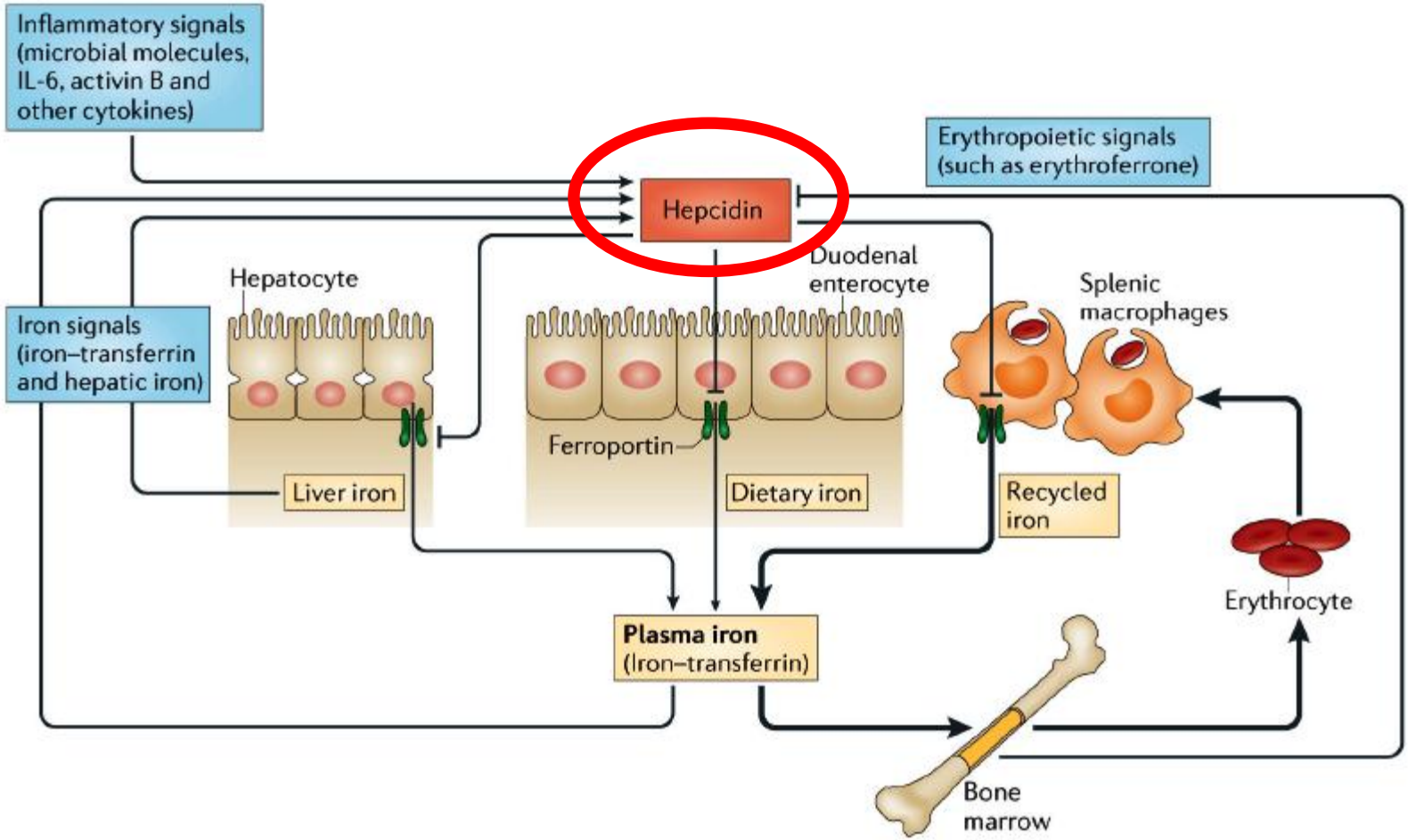
Iron homeostasis in host defence and inflammation

Tomas Ganz[1,2] and **Elizabeta Nemeth**[1]

[1]Department of Medicine, David Geffen School of Medicine at University of California, Los Angeles, California 90095–1690, USA

[2]Department of Pathology, David Geffen School of Medicine at University of California, Los Angeles, California 90095–1690, USA





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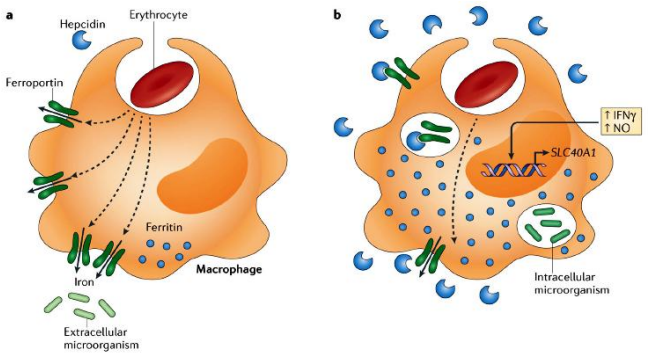

The increase in hepcidin secretion in response to infectious or inflammatory stimuli is responsible for the characteristic hypoferraemia of inflammation[26,27], which develops within a few hours of a systemic infection or following injection of microbial components or certain cytokines. High hepcidin concentrations cause iron sequestration in macrophages, which effectively redistributes iron not only from the plasma compartment but also from non-macrophage storage in hepatocytes. Prolonged activation of this mechanism during infection and inflammation imposes a biological cost in the form of anaemia of inflammation[27–29], a condition that is partly caused by the restriction of iron supply to the erythropoietic bone marrow.

International Journal of Laboratory Hematology
The Official journal of the International Society for Laboratory Hematology
REVIEW ARTICLE INTERNATIONAL JOURNAL OF LABORATORY HEMATOLOGY

The mutual control of iron and erythropoiesis

C. CAMASCHELLA, A. PAGANI, A. NAI, L. SILVESTRI

La sideropenia, intesa come riduzione del ferro circolante è quindi considerata un meccanismo protettivo di fronte al pericolo rappresentato dall'invasione di microrganismi e il fatto che la carenza di ferro causi anemia, rientra nella strategia del "male minore". Non solo il ferro è sequestrato dall'incremento di epcidina ma nell'infiammazione aumentano anche la concentrazione di aptoglobina ed emopessina che rimuovono dalla circolazione, rispettivamente, emoglobina o eme in eccesso attraverso specifici recettori scavenger come il CD163 dei macrofagi, pure attivato nella infiammazione. Tutto ciò al fine di non peggiorare il danno tissutale già causato dall'infiammazione/infezione e di recuperare il ferro legato all'eme.

Anemia and Transfusion in Critically Ill Pediatric Patients

A Review of Etiology, Management, and Outcomes

Daniel Sloniewsky, MD

However, over the last 20 years it has become evident that transfused blood does not necessarily demonstrate the same characteristics as autologous blood. In fact, transfused blood products demonstrate metabolic, physiologic, and immunomodulatory effects that may worsen patient outcomes, possibly because of problems associated with the storage process. Subsequently multiple clinical studies have

Iron-deficiency anemia caused by increased blood loss or inadequate dietary intake can also occur in critically ill patients. The diagnosis of total body iron depletion, however, can be complicated by the presence of anemia of inflammation, because the usual markers of iron deficiency are themselves affected by inflammation. Novel indicators such as Erythrocyte Zinc Protoporphyrin and Transferrin Receptor levels have been proposed as potentially useful in the diagnosis of iron deficiency.[10]

Crit Care Clin 29 (2013) 301–317

Anemia and Transfusion in Critically Ill Pediatric Patients

A Review of Etiology, Management, and Outcomes

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Transfusion-associated circulatory overload

Transfusion-associated circulatory overload (TACO), although not an immunologic phenomenon, is mentioned here because it can also present as pulmonary edema and, therefore, may be difficult to discern from TRALI. Its incidence has been quoted as from 1% to 10%, but is difficult to truly know because of the lack of an accepted definition. However, unlike TRALI, the etiology of TACO is thought to be related to an increase in hydrostatic pressure in the pulmonary vasculature, and patients with cardiopulmonary compromise and/or renal failure are at higher risk, as are infants.[54] Just as for TRALI there is no definitive test to diagnose TACO, although echocardiography, B-type natriuretic peptide level, and alveolar fluid protein content can be helpful. Because it can be somewhat prevented by using slow infusion rates in at-risk patients and does respond to diuretic therapy, there is a benefit in a diagnosis of TACO rather than TRALI.[49]

Iron supplementation can be administered via enteral or intravenous routes. The enteral route seems to be ineffective, as absorption may be downregulated in patients with increased iron stores.[75] Enteral iron also requires an acidic environment to be absorbed, which may be hindered by medications commonly given to critically ill patients to suppress secretion of gastric acid. Pieracci and Barie[12] demonstrated

Crit Care Clin 29 (2013) 301–317

EAST 2015 PLENARY PAPER

Chronic restraint stress after injury and shock is associated with persistent anemia despite prolonged elevation in erythropoietin levels

Letitia E. Bible, MD, Latha V. Pasupuleti, MD, Amy V. Gore, MD, Ziad C. Sifri, MD, Kolenkode B. Kannan, PhD, *and* **Alicia M. Mohr, MD,** *Gainesville, Florida*

Chronic Stress Model

Following LC or LCHS, everyday between 8:00 AM and noon, animals underwent restraint stress. Animals were placed in restraint containers, which measured 16.5 cm in length and 7.5 cm in diameter, for 2 hours daily. To prevent acclimation to containers, animals were stimulated at 30, 60, and 90 minutes. Stimulation consisted of 2 minutes of continuous alarms (80–85 dB) transmitted via speakers placed adjacent to restraint containers as well as repositioning. Animals were removed from their restraint containers at 2 hours and returned to normal housing in 12-hour light/dark cycles. Since animals undergoing CS were not permitted feed or water, animals in all other experimental groups were not given access to feed or water during this 2-hour period.

J Trauma Acute Care Surg
Volume 79, Number 1

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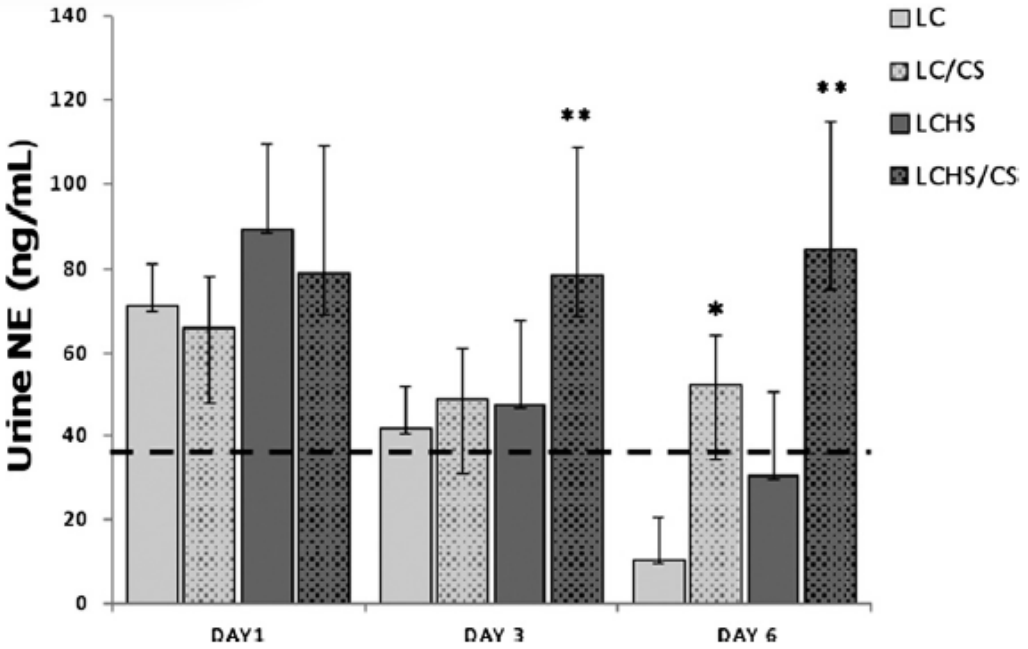

Figure 1. Urinary NE level 1, 3, and 6 days after injury, shock, and CS. On Day 1, all groups have elevated urinary NE levels compared with the control animals (represented by the *dashed line*). The addition of CS to LC and LCHS results in significantly increased urinary NE levels on Day 6. LC, LC alone; LC/CS, LC followed by SC. *$p < 0.05$ versus LC alone. **$p < 0.05$ versus LCHS alone. *Dashed line* represents control level.

Chronic restraint stress after injury and shock is associated with persistent anemia despite prolonged elevation in erythropoietin levels

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In summary, the results of this study implicate CS as a cause of persistent NE elevation that is associated with prolonged BM dysfunction and anemia seen following severe traumatic injury. The addition of CS to either LC or LCHS results in a prolonged decrease in BM cellularity and BM HPC growth. This BM dysfunction is associated with a prolonged anemia despite significant elevation in circulating EPO levels. Chronic restraint stress following injury and shock provides a clinically relevant model to further evaluate persistent injury-associated anemia, which mimics critical illness seen in trauma ICU patients.

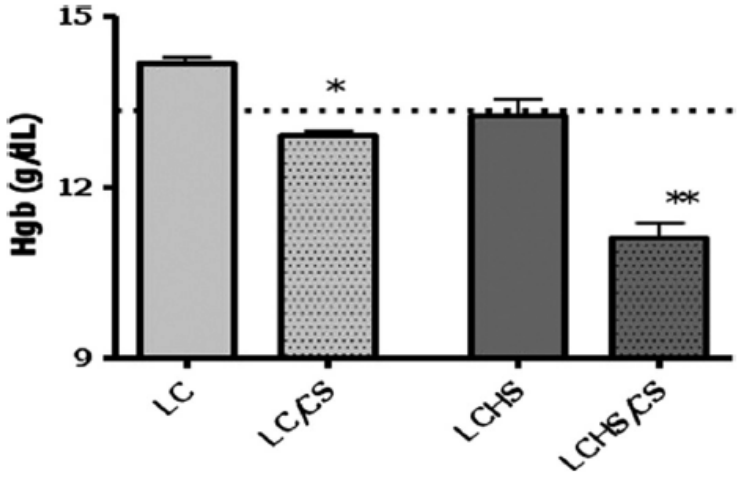


Figure 3. Hgb 7 days after injury, shock, and CS. Hgb after 7 days of CS is also only slightly decreased from control Hgb at 7 days after LCHS approaches control levels. The addition of SC after LCHS causes a significant reduction of Hgb at 7 days. LC, LC alone; LC/CS, LC followed by CS; LCHS, LC followed by hemorrhagic shock; LCHS/CS, LCHS followed by CS. *$p < 0.01$ versus LC alone. **$p < 0.001$ LCHS. *Dashed line* represents control levels.

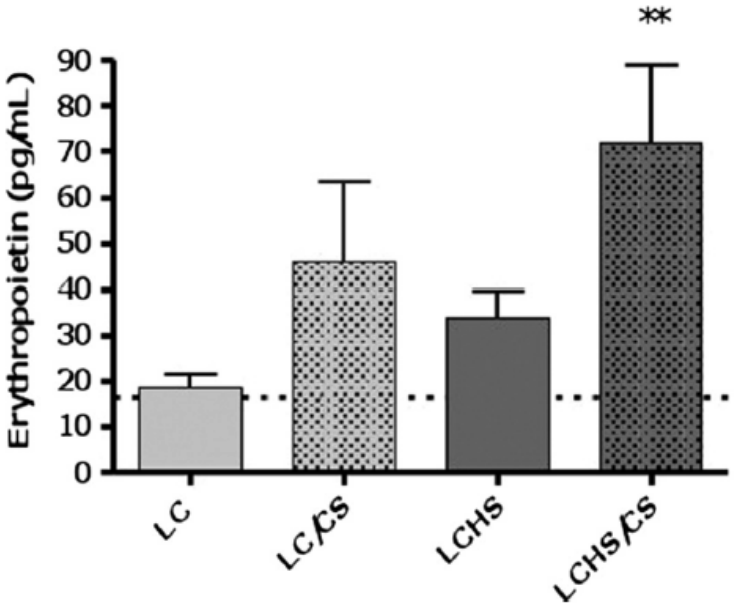


Figure 4. EPO levels 7 days after injury, shock, and CS. The addition of CS to LC and LCHS results in increased EPO levels. LC, LC alone; LC/CS, LC followed by CS; LCHS, LC followed by CS. **$p < 0.05$ versus LCHS alone. *Dashed line* represents control level.

Nutrición Hospitalaria

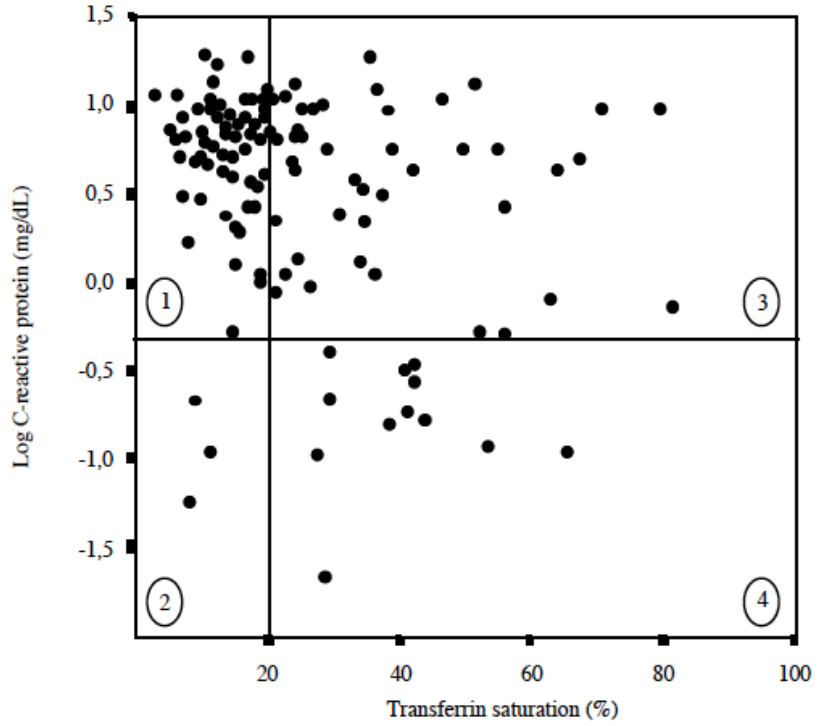
Nutr. Hosp. (2005) XX (2) 115-120
ISSN 0212-1611 • CODEN NUHOEQ
S.V.R. 318

Original

Iron metabolism, inflammation and anemia in critically ill patients. A cross-sectional study

M. Muñoz, A. Romero*, M. Morales**, A. Campos*, J. A. García-Erce*** y G. Ramírez*

*GIEMSA: Department of Biochemistry and Molecular Biology. School of Medicine. *Departments of Haematology and **Clinical Biochemistry. University Hospital "Virgen de la Victoria". University of Málaga. Spain. ***Department of Haematology. University Hospital "Miguel Servet". Zaragoza. Spain.*



1. CRP > 0.5 mg/dL and SatTRF < 20% (n = 66): Functional iron deficiency (FID) alone[42] or with real iron deficiency (sTfrR > 2.3, n = 14).
2. CRP < 0.5 mg/dL and SatTFR < 20% (n = 3): Real iron deficiency.
3. CRP > 0.5 mg/dL and SatTFR > 20% (n = 50): Inflammation without FID.
4. CRP < 0.5 mg/dL and SatTFR > 20% (n = 12): Normal.

Fig. 1.—Correlation between levels of inflammatory marker (Log C-reactive protein) and functional iron deficiency (% transferring saturation) in 131 ICU patients.

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Results: **Anaemia (Hb < 12 g/dL) was present in 76% of the patients (Hb < 10 g/dL in 33%), hypoferremia (SI < 45 μg/dl) in 69%; satTRF < 20% in 53%; FRT < 100 ng/mL in 23%; sTfR > 2.3 mg/dL in 13%, and CRP > 0.5 mg/dL in 88%. Statistically significant correlations (r of Pearson; *p < 0.05, **p < 0.01) were obtained for serum CRP levels and WBC**, Hb*, TRF**, satTRF*, and FRT**. There was also a strong correlation between TRF and FRT (-0.650**), but not between FRT and satTRF or SI. LIS correlated with Hb*, CRP**, TRF*, satTRF* and FRT**.**

Conclusion: **A large proportion of critically ill patients admitted to the ICU presented the typical functional iron deficiency (FID) of acute inflammation-related anaemia (AIRA). This FID correlates with the inflammatory status and the length of stay at the ICU. However, 21% of the ICU patients with AIRA had an associated real iron deficiency (satTRF < 20; FRT < 100 and sTfR > 2,3). Since oral supplementation of iron seems to be ineffective, all these patients might benefit of iv iron therapy for correction of real or functional iron deficiency, which in turn might help to ameliorate their inflammatory status.**

Diagnosi!

La ferritina non serve!

Sideremia / (Transferrina x 1,42) x 100

Valori normali 15-45%

ORIGINAL ARTICLE

A fast-track anaemia clinic in the Emergency Department: feasibility and efficacy of intravenous iron administration for treating sub-acute iron deficiency anaemia

Manuel Quintana-Díaz[1,2,3], Sara Fabra-Cadenas[1,3], Susana Gómez-Ramírez[4], Ana Martínez-Virto[1,3], José A. García-Erce[3,5], Manuel Muñoz[4]

[1]Emergency Department; [2]Intensive Care Unit, University Hospital La Paz, Madrid; [3]Emergency Medicine Research Group, Research Institute University Hospital La Paz (IdiPAZ), Madrid; [4]Transfusion Medicine, School of Medicine, University of Málaga, Málaga; [5]Haematology and Haemotherapy Service, General Hospital San Jorge, Huesca, Spain

Table I - Indications for transfusion according to patients' characteristics.

Patients' characteristics	Transfusion trigger*
Sub-acute anaemia in asymptomatic patients	Hb<5 g/dL
Sub-acute anaemia in young patients with clinical signs/symptoms and without risk criteria**	Hb<6 g/dL
Sub-acute anaemia in elderly patients with clinical signs/symptoms and without risk criteria	Hb<7 g/dL
Sub-acute anaemia in patients with risk criteria and without clinical signs/symptoms	Hb<8 g/dL
Sub-acute anaemia in patients with clinical signs/ symptoms and risk criteria	Hb<9 g/dL

*Adapted from recommendations of the Spanish Society of Blood Transfusion and Cellular Therapy[35]; **Risk criteria: coronary artery disease/ cardiac valve disease, heart failure, cerebrovascular disease or obstructive pulmonary disease. Hb: haemoglobin.

Anaemia classification

The type of anaemia was classified within 7 days in the fast-track anaemia clinic. Anaemia with absolute iron deficiency was defined by ferritin <30 ng/mL (or <100 ng/mL if TSI<20%); functional iron deficiency by TSI<20% and ferritin >100 ng/mL (anaemia of chronic inflammation); vitamin B_{12} deficiency by a serum level <200 pg/mL; and folic acid deficiency by a serum level <5.4 ng/mL.

*Blood Transfus 2016; **14:** 126-33* DOI 10.2450/2015.0176-15

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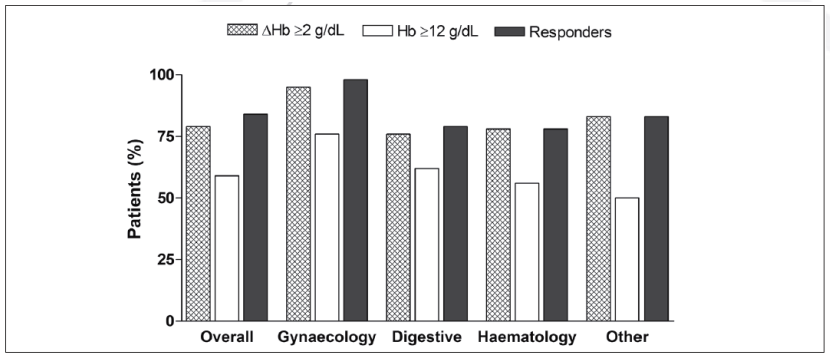

Figure 2 - Percentage of patients showing a haematological response (responders), as defined by a haemoglobin increment (ΔHb) ≥2 g/dL and/or a haemoglobin level ≥12 g/dL, at 4 weeks after administration of the total dose of iron.

Conclusions

In summary, although some of the anaemic patients attending an ED may need RBC transfusion for conditions such as acute anaemia with haemodynamic instability, most of them present with chronic iron deficiency anaemia and could benefit from optimisation of their Hb level and iron stores with IV iron. Unfortunately, they still receive RBC transfusion because it is readily available and erroneously considered a safe and inexpensive therapeutic option. Our data seem to support - to the best of our knowledge, for the first time - the implementation of a fast-track anaemia clinic in the ED for management of patients presenting with sub-acute, moderate-to-severe anaemia, and the administration of FCM as a safe, durable and probably cost-effective option for iron supplementation in such patients. Early diagnosis and treatment of iron deficiency anaemia in the ED will most probably result in improved use of blood products in this hospital healthcare area.

*Blood Transfus 2016; **14:** 126-33* DOI 10.2450/2015.0176-15

Dottò, i miei figli non sanno che io ho capito so che sto per morire.
Vi prego non mi fate morire in ospedale.
Abito a Coroglio, di fronte al mare.
La mattina vedo sorgere il sole appena dietro l'isola di Nisida.
Bennato dice che Nisida è un'isola e nessuno lo sa.
Per me è un sipario su una meravigliosa alba!
Vi prego mandatemi a casa è l'ora del tramonto.

CHIMICA CLINICA - IMMUNOMETRIA

Cod. struttura 4

LAMORGESE PASQUALE
VIA SAN DOMENICO 18
80100 NAPOLI (NA)
Data di nascita : 22/02/1928
(3317) (30) (*)

NAPOLI, prelievo del 02/03/2016

determinazioni richieste	esiti		valori di riferimento (Uomo)
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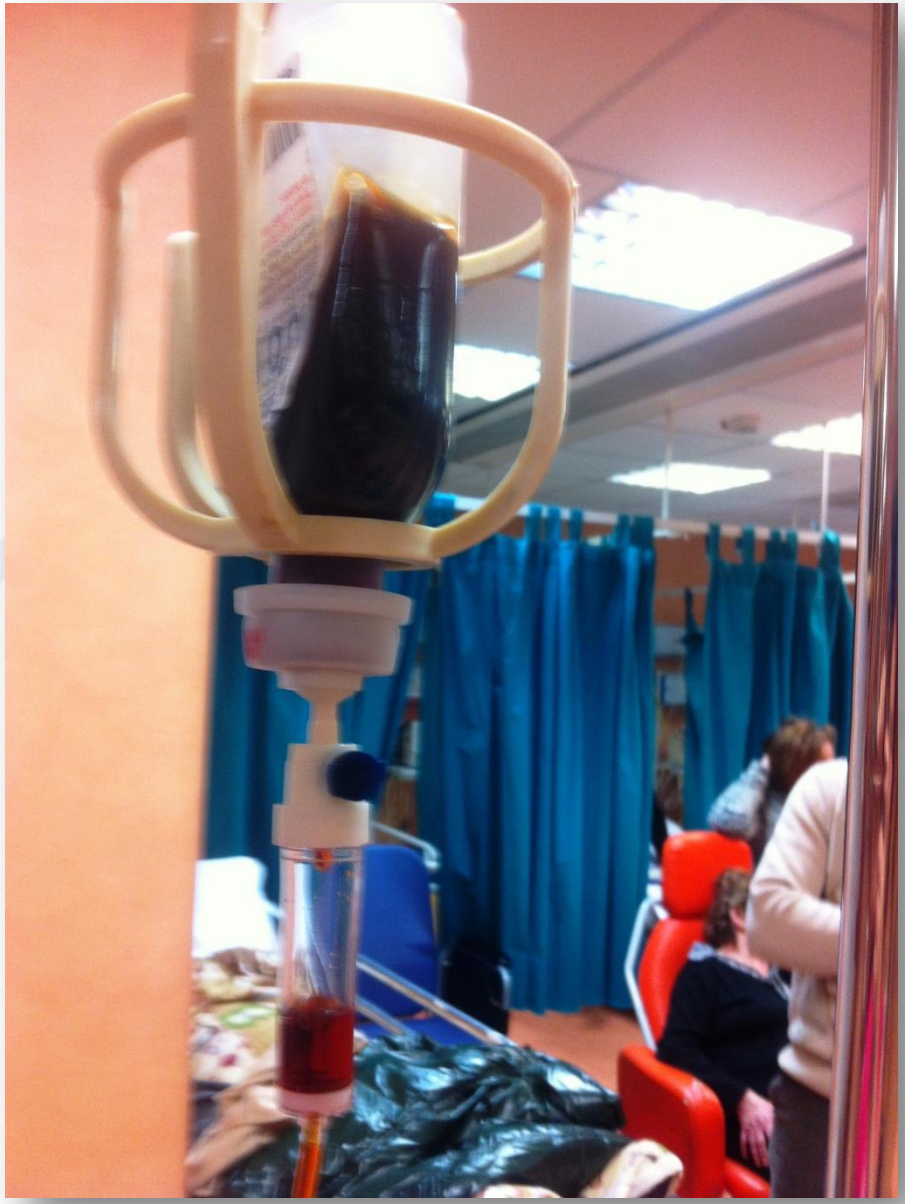
TRANSFERRINEMIA **Nefelometria**	**103**	**mg/dl**	200 - 360

Sat. Transferrina 8%

Il Direttore Tecnico
Dott. Claudia Marengo



a : Responsabilita di Terzi SI ☐ NO ☐ Omissione di soccorso: SI ☐ NO ☐

cidente :

ircostanze dichiarate:

RA - 02/03/2016 ore 15:24 Note: ESEGUI

RA - 02/03/2016 ore 18:34

IMISSIONE

ANEMIA SIDEROPENICA SEVERA. SI CONSIGLIA TERAPI AMARZIALE CON FERRO CARBOSSI-MALTOSIO.

5:14 **Parametri vitali** P.A.(Max/Min): 136/84 mm/Hg F.C.: 98 bmp SPO2: 95 Note: PRATICATO ECG

RELLI LUCIA dal 02/03/2016 15:24

5:24 **Laboratorio** UREA,TEMPO DI PROTROMBINA,SODIO,POTASSIO,GLUCOSIO,EMOCROMO,CREATININA,CK-MB,CPK,BILIRUBINA TOTALE,BILIRUBINA DIRETTA,TROPONINA I

:58 **Esame Obiettivo** Il paziente viene in PS per anemia severa. K vescicale con localizzazioni secondarie. IRC di grado severo. iponatriemia. Condizioni cliniche scadute Cuore: Toni chiari e pause libere Si pratica counseling al figlio per il bilancio rischi benefici della terapia trasfusionale con emazie concentrate (rischio di sovraccarico di volume oltre al rischio legato all'emotrasfusione) versus terapia marziale con ferro carbossimaltosio. Ferritinemia non utilizzabile per l'inquadramento del paziente per l'iperspressione dell'Epcidina da processo infiammatorio-oncologico. Sideremia 13 Trasferrinemia 103 Percentuale di Saturazione della Trasferrinemia 8& (v.n. >15%). Ferrinject 500 2 fiale in fisiologic 250 ml a 250 ml/ora Si pianifica accesso programmato in Medicina Generale per proseguire la terapia marziale con ferro carbossimaltosio, martedì mattina con impegnativa del medico curante indicante la dicitura "Anemia severa in paziente affetto da insufficienza renale cronica e K-vescicale. Ricovero in day-hospital per terapia marziale infusiva co ferro-carbossimaltosio. Iponatremia: NaCl ipertonico 2 fiale in fisiologica 250 ml

4 **Esame Obiettivo** Infusione di Ferro e di NaCl ipertonico avvenuta senza esiti nè complicanze

REGIONE CAMPANIA - A.S.L NA 1 CENTRO - OSPEDALE "SAN PAOLO"
Unità Operativa Complessa di MEDICINA DI LABORATORIO
Responsabile: Prof. Flavia Ingala
Via Terracina n. 219 - Napoli - Tel. (081) 2547859 - 2547866 Fax (081) 2547865
Indirizzo e-mail : lab.s.paolo@libero.it

Cod. : **11399**
Provenienza: **Medicina Generale**

Accettato il : **22/03/2016**
Refertato il : **22/03/2016**

Sig. **LAMORGESE PASQUALE**
C.F. **LMRPQL28B22F839U**
D.Nasc. **22/02/1928**
DH

ESAME	RISULTATO	UNITA'	RIFERIMENTO

ESAME EMOCROMOCITOMETRICO

WBC	**11,2** *	$10^3/mm^3$	4.2 - 10.5
RBC	* **3,35**	$10^6/mm^3$	4.0 - 5.8
HGB	* **9,4**	g/dl	11.5 -17.5
HCT	* **30,7**	%	36.0 - 55.0
MCV	**92,0**	μm^3	78,0 - 98,0
MCH	**28,1**	pg	27,0 - 34,0
MCHC	* **30,7**	g/dl	32,0 - 36,0
RDW	**19,2** *	%	11,0 - 18,0
PLT	**471** *	$10^3/mm^3$	150 - 400

FORMULA LEUCOCITARIA

	%		$10^3/mm^3$	
LYN	* **13,1**	19,0 - 48,0	**1,47**	1.0 - 4.8
MON	**3,5**	1,0 - 10,0	**0,39**	0.1 - 0.8
NEU	**82,5** *	40,0 - 75,0	**9,24** *	1,8 - 7,0
EOS	**0,8**	0,0 - 6,0	* **0,09**	0,1 - 0.44
BAS	**0,1**	0,0 - 1,5	**0,01**	0,0 - 0.20

fine referto

IL REFERTO HA VALORE MEDICO-LEGALE SOLTANTO SE TIMBRATO E FIRMATO DAL SANITARIO DEL LABORATOR
N.B. TRASMISSIONE TELEMATICA DEL REFERTO IN VIA SPERIMENTALE.

REGIONE CAMPANIA - A.S.L NA 1 CENTRO - OSPEDALE "SAN PAOLO"
Unità Operativa Complessa di MEDICINA DI LABORATORIO
Responsabile: Prof. Flavia Ingala
Via Terracina n. 219 - Napoli - Tel. (081) 2547859 - 2547866 Fax (081) 2547865
Indirizzo e-mail : lab.s.paolo@libero.it

Cod. : **11399**
Provenienza: **Medicina Generale**

Accettato il : **22/03/2016**
Refertato il : **22/03/2016**

Sig. **LAMORGESE PASQUALE**
C.F. **LMRPQL28B22F839U**
D.Nasc. **22/02/1928**
DH

ESAME	RISULTATO	UNITA'	RIFERIMENTO
UREA	**61** *	mg/dl	10-50
GLUCOSIO	**100**	mg/dl	70 - 110
CREATININA	**2,99** *	mg/dl	0.50 - 1.17
SODIO	* **126**	mEq/l	135-155
POTASSIO	**5,3**	mEq/l	3.5-5.5
CLORO	* **90**	mEq/l	96-111
FERRO	* **26**	µg/dl	59 - 158
TRANSFERRINA	* **92**	mg/dl	200 - 360

fine referto

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Cod. : **11399**
Provenienza: **Medicina Generale**

Accettato il : **22/03/2016**
Refertato il : **24/03/2016**

Sig. **LAMORGESE PASQUALE**
C.F. **LMRPQL28B22F839U**
D.Nasc. **22/02/1928**
DH

ESAME	RISULTATO	UNITA'	RIFERIMENTO
FERRITINA	**1353,0** *	ng/ml	uomini 18 - 341

fine referto

IL SANITARIO RESPONSABILE

IFERTO HA VALORE MEDICO-LEGALE SOLTANTO SE TIMBRATO E FIRMATO DAL SANITARIO DEL LABORATORIO.
TRASMISSIONE TELEMATICA DEL REFERTO IN VIA SPERIMENTALE.

IL SANITARIO RESPONSA

GALE SOLTANTO SE TIMBRATO E FIRMATO DAL SANITARIO DEL LABORATORIO.